0.21-0.79; p=0.0075). After multivariable analysis, the management during the period 2 remained a strong protective factor (adjusted hazard ratio=0.26; 95% confidence interval, 0.09-0.76; p=0.014). During period 2, we observed a significant better compliance in antimicrobial therapy and fewer cases of renal failure. Deaths by embolic events and multiple organ failure syndrome also significantly decreased during period 2.

Conclusions: A dramatic reduction in mortality was observed during this study suggesting that a management-based approach has a significant impact on IE outcome.

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Predictors of mitral valve replacement after percutaneous mitral valvuloplasty

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Introduction: Percutaneous mitral valvuloplasty (PVM) is an effective therapy for mitral stenosis in selected patients. Nonetheless, mitral valve replacement (MVR) may be still required after PVM in some cases. The aim of this analysis is to assess predictors of MVR after PVM.

Methods: Our retrospective study includes 354 patients enrolled in our department (1996-2002).

Results: Thirty seven (10, 34%) of 354 patients underwent MVR at a mean interval of 13,6±9,3 months after PVM. No major differences were apparent in the demographic features of patients who did or did not undergo surgery. Five patients (13%) underwent surgery within the first month after PVM while 32 patients (87%) were operated later. On univariate analysis several predictors were brought out: rheumatic antecedents, commissurotomy history, atrial fibrillation, severe mitral regurgitation before PVM or its aggravation after PVM, high echocardiography score, calcification, small mitral area, mitral gradient, diastolic and systolic arterial pulmonary pressure before PVM. However, on multivariate analysis only mitral area, diastolic and systolic arterial pulmonary pressure could be predictors for surgery.

Conclusion: Certainly, PVM is an excellent treatment option in mitral stenosis. It is minimally invasive, well-tolerated and has a high success rate. But the need of surgery after PVM is not uncommon, so if a mitral valve restenosis occurred in patients with predictors of surgery, do we repeat PVM or directly perform RMV?

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History of stroke in patients with paroxysmal supraventricular tachycardia. clinical significance

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Atrial fibrillation (AF) is a major cause of embolic event, but the significance of other arrhythmias remains unknown. The purpose of study was to assess the significance of unexplained stroke in patients who had apparently only supraventricular tachycardia (SVT).

Population: Unexplained stroke was noted in 26 patients (pts) (group I) admitted for palpitations and electrophysiological study (EPS); they were issued from a population of 1002 pts with normal ECG in sinus rhythm, palpitations or tachycardia and with inducible SVT at EPS; 976 pts had no history of stroke (group II).

Methods: EPS consisted of atrial pacing and programmed atrial stimulation in control state and if necessary after infusion of isoproterenol. Clinical and electrophysiological data of both groups were studied. Pts with stroke had a normal carotid ultrasound study and transcranial Doppler ultrasonography.

Results: Pts with unexplained stroke represent 2.5 % of population with SVT. They were older than remaining pts without stroke (65±10 vs 50±19 years) (p 0.001). They have more frequently associated heart disease (8/26, 31 % vs 37/976, 4 %) (p < 0.001). Male gender was more frequent in group I (16/26, 60 %) than in group II (355/956, 37 %) (p< 0.05). Typical AV node re-entrant tachycardia was noted in 17 group I pts (65 %) and 693/976 (68 %) (NS); AV re-entrant tachycardia associated with a concealed accessory pathway was noted in 2 group I pts (8 %) and 168 group I pts (17 %) (NS); atypical AV node re-entrant tachycardia was more frequent in group I (7/26, 27 %) than in group II (115/976, 12 %) (p<0.05). AF induction was more frequent in group I (6/26, 23 %) than in group II (35/976, 3.5 %) (p< 0.001). Ablation was performed in 6 group I pts; one of them died from another stroke; another one died suddenly; another one developed a permanent AF.

Conclusions: Old age, male gender, associated heart disease, and induction of atrial fibrillation were significant factors associated with the history of stroke in pts with SVT. They have a risk of adverse events during the follow-up. However, unexplained stroke was a rare event in patients with paroxysmal SVT, noted in 2.5 % of this population.

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Determinants of Plasma Brain Natriuretic Peptide Release in Aortic Stenosis

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Introduction: Brain natriuretic peptide (BNP) is released by the left ventricle (LV) in response to increase LV wall stress and is associated with symptoms and outcome in aortic stenosis (AS). However, the relation between BNP, LV filling pressure and left atrial (LA) volume has been poorly examined in AS. This study was undertaken to assess the determinants of BNP level in AS.

Method and results: Two-dimensional, tissue Doppler imaging and Doppler transthoracic echocardiography were performed in 34 consecutive patients with severe AS (72±12 years, 62% of male, aortic valve area (AVA), 0.67±0.2 cm^2, mean gradient, 45±13mmHg). Concomitantly, plasma BNP level was measured. Mean plasma level BNP was 325±572pg/ml. BNP was significantly higher in patients with reduced LV systolic function (LV ejection fraction ≤ 60%, n=22; 595±869 vs. 178±235pg/ml, p=0.04), increased indexed LA volume (≥ 40ml/m^2, n=21; 454±686 vs. 125±210pg/ml, p=0.026) and increased LV filling pressure (E/Ea ratios: septal ≥ 15, n=17, 562±738 vs. 108±117pg/ml, p=0.004; lateral ≥ 11, n=14, 626±800 vs. 137±139, p=0.02). There were good correlations between BNP and LV and LA ejection fraction, (r=-0.40, p=0.0035 and r=-0.45, p=0.01), indexed LA volume (r=0.48, p=0.0049) and with septal and lateral E/Ea ratios (r=0.69, p<0.0001 and r=0.50, p=0.0054). However, there was lower correlation between BNP and AVA (r=0.33, p=0.047). After adjustment for age and LV ejection fraction, multivariate analysis showed that E/Ea septal (p=0.0005) or lateral (p=0.048) ratios and indexed LA volumes (p=0.006) remained associated with BNP. Furthermore, symptomatic patients (n=24) had significantly higher BNP level (427±654 vs.78±112pg/ml, p=0.009) and E/Ea septal (25±14 vs. 12±7, p=0.01) and lateral ratios (15±7 vs. 10±5, p=0.01) than asymptomatic patients.

Conclusion: This study shows that BNP release is mainly determined by both LV and LA function in patients with AS. In this population, BNP level reflects the ventricular and atrial consequences of increased afterload rather than the severity of stenosis.